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Claims:

1. A method of generating an immune response in a patient to an antigen in a patient comprising:

administering to the patient an immunoglobulin or portion thereof wherein said immunoglobulin has at least one peptide epitope of said antigen attached to said immunoglobulin or portion thereof and administering said immunoglobulin or portion thereof in conjunction with a RNA segment.

2. The method of claim 1 wherein the immunoglobulin or portion thereof and said RNA segment are administered together.

3. The method of claim 1 wherein the immunoglobulin or portion thereof and said RNA segment are administered separately.

4. The method of claim 1 wherein said patient is human.

5. The method of claim 1 wherein upon administration of said immunoglobulin or portion thereof to said patient the immunoglobulin or portion thereof loads the antigen presenting cell by engagement with the antigen presenting cell's FcγR and said peptide epitope is effectively processed and presented by the MHC I pathway of the antigen presenting cell resulting in effective loading of the MHC class I molecules.

6. The method of claim 1 wherein the peptide epitope is attached within the CDR region of the immunoglobulin or portion thereof.

7. The method of claim 1 wherein the immune response generates an effective T cell response to the antigen.

8. The method of claim 7 wherein the T cells are cytotoxic T lymphocytes.

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9. The method of claim 1 wherein the RNA segment is dsRNA and is selected from the group consisting of pA:pU and pI:pC.

10. The method of claim 1 wherein the peptide epitope is a T cell epitope.

11. The method of claim wherein the peptide epitope is selected from the group consisting of influenza virus M1 or M2; hepatitis C virus NS3; hepatitis B virus core antigen; human papilloma virus HPV 18-E7, HPV 16 – E7, HPV 18 E6, HPV 16 E6; melanoma –gp100; MART-1; TRP-2; carcinoembryonic antigen precursor; Her –2; tetanus toxin universal T helper epitope; HIV-1: reverse transcriptase; HIV1: gag; insulin precursor – human; human Gad 65; prostate tumor antigens; mucin 1; herpes simplex antigens; and, respiratory syncytial virus antigens.

12. The method of claim 1 wherein the immunoglobulin or portion thereof and RNA segment is administered by one of the methods selected from the group consisting of intravenous administration and bolus injection.

13. The method of claims 1 wherein the immunoglobulin or portion thereof and RNA are administered in a pharmaceutically acceptable carrier.

14. The method of claim 1 wherein the method induces an effective memory response to the peptide epitope.

15. A method of loading an antigen presenting cell and generating a T cell response by use of at least one peptide epitope attached to an Ig backbone or portion thereof thereby forming an Ig –peptide molecule wherein when administered *in vivo* the epitope is effectively processed and presented by the MHC I pathway resulting in effective loading of MHC class I molecules thereby resulting in an MHC class I – peptide complex.

16. The method of claim 15 wherein the Ig backbone is an IgG backbone.

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17. The method of claim 15 wherein the APC is loaded via monovalent engagement of FcγR.

18. The method of claim 15 wherein the APC may be loaded *in vivo* or *ex vivo*.

19. The method of claim 15 wherein the peptide epitopes are covalently attached to the IgG backbone.

20. The method of claim 15 wherein the peptide epitope is attached to the IgG backbone without modification of the Fc portion of the IgG.

21.The method of claim 15 wherein the MHC class I –peptide complex results in generation of robust Tc2 responses characterized by IL-4 but not IL-2 or IFN-γ- production.

22. The method of claim 15 wherein the peptide epitope is selected from the group consisting of influenza virus M1 or M2; hepatitis C virus NS3; hepatitis B virus core antigen; human papilloma virus HPV 18-E7, HPV 16 – E7, HPV 18 E6, HPV 16 E6; melanoma –gp100; MART-1; TRP-2; carcinoembryonic antigen precursor; Her –2; tetanus toxin universal T helper epitope; HIV-1: reverse transcriptase; HIV1: gag; insulin precursor – human; human Gad 65; prostate tumor antigens; mucin 1; herpes simplex antigens; and, respiratory syncytial virus antigens.

23. The method of claim 15 wherein the negative effects of sera are avoided.

24. The method of claim 15 wherein the method results in the formation of MHC-peptide complexes.

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25. The method of claim 15 wherein the IgG peptide molecule is administered by subcutaneous or intraperitoneal injection.

26. The method of claim 15 wherein the antigen presenting cell is selected from the group consisting of dendritic cells, monocytes, macrophages and B cells.

27. The method of claim 15 wherein the antigen presenting cell is selected from the group consisting of CD11c+ and CD11b+ APC.

28. The method of claim 15 wherein the resulting MHC–peptide complexes formed by *in vivo* delivery are expressed for up to 1 to 2 weeks.

29. The method of claim 15 wherein the MHC-peptide complex results in activation of T cells.

30. The method of claim 29 wherein the T cell response is determined by ITAM+ and ITIM+ Fcgamma receptors on APC.

31. The method of claim 30 wherein expression of the gamma chain of ITAM+ FcγR isoforms induces the T cell response wherein ITIM+ FcγRII limits the T cell response.

32. The method of claim 15 wherein when APC are loaded *in vivo* and the APC induce distinct regulatory subsets.

33. The method of claim 26 wherein monocytes induce Th2 and Tr1 cells, both dendritic cells and monocytes induce Th3 cells, and wherein CD11b+ monocytes are more potent than dendritic cells in triggering a regulatory response following IgG-mediated delivery of T cell epitope.

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34. The method of claim 15 wherein the loading of APC with a peptide delivered within an IgG backbone *in vivo* results in induction of Th2 immunity.

35. The method of claim 15 wherein the loading of APC with a peptide delivered within an IgG backbone *in vivo* results in induction of Th3 and Tr1 immunity.

36. The method of claim 15 wherein the T cell response is enhanced by co-stimulation with one of the following selected from the group consisting of anti-CD40mAb, recombinant IL-12 or synthetic dsRNA.

37. The method of claim 15 wherein IL-2, IFN-γ and IL-4, were down-regulated in a dose dependent manner and IL-10 and TGF-beta were upregulated in a dose-dependent manner.

38. The method of claim 15 wherein the peptide epitope is recNP and induced NP-specific MHC class I-restricted T cell immunity consisting of IL-4 producing Tc2 cells.

39. The method of claim 15 further comprising the use of RNA motifs thereby resulting in an enhanced immune response.

40. The method of claim 39 wherein the RNA motifs are dsRNA.

41. The method of claim 39 wherein the IgG1 and IgG2a antibody responses were increased and associated with an enhanced Th1 and Th2 response.

42. The method of claim 40 wherein the dsRNA was selected from the group consisting of pA:pU, pI:pC and pC:pG.

43. The method of claim 39 wherein the dsRNA is pA:pU and induced MHC class I-restricted Tc1 cells thereby producing IFN-γ.

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44. The method of claim 40 wherein the dsRNA are from 10 - 50Kd.

45. The method of claim 39 wherein the RNA motifs are ssRNA selected from the group consisting of p(A), p(C), p(G), p(I) and p(U).